Dairy Consumption Reduces Risk of Kidney Disease

- Habitual intake of low- or reduced-fat dairy products is associated with a lower risk for developing chronic kidney disease.
- Dairy foods may influence kidney function because they lower the risk for developing hypertension and type 2 diabetes, the two leading causes for chronic kidney disease.
- Dairy components, including calcium, magnesium, phosphorus, and protein, may directly influence kidney function by modifying inflammation and oxidative stress.

Dairy foods are best known for promoting a healthy skeleton, but bones are not the only tissue to reap their health benefits. The very same dairy ingredients—calcium, magnesium, phosphorus, potassium, and protein—that build and maintain a healthy skeleton have demonstrated protective effects on cardiovascular disease, type 2 diabetes (T2D), and hypertension [1]. And now a growing body of evidence [1-6] suggests habitual dairy consumption may benefit the kidneys as well.

Chronic kidney disease (CKD) currently affects 27 million adults in the U.S. [7] and nearly 10% of adults worldwide [8]. CKD is diagnosed based on a filtration rate—the glomerular filtration rate (GFR) to be exact—because the kidneys’ job is to filter the blood. Normal physiological functions, such as protein digestion and muscle activity, create waste products (e.g., urea, creatinine) that need to be removed from the blood and passed into urine. The GFR measures how much of this waste the body filters in a given amount of time. A normal GFR is between 90 and 120 milliliters of blood per minute [1, 7]. Although there is variation in what is considered normal with respect to age, sex, ethnicity, and body size, values below 90 ml/min are considered indicative of abnormal kidney function [7]. CKD is diagnosed when a patient has a GFR less than 60 ml/min for three or more months [1, 5, 7, 8].

The link between dairy foods and bone health is apparent; milk, cheese, and yogurt supply the very minerals needed for bone health. Calcium, phosphorus, and magnesium are all present in dairy products as a natural part of the milk's structure. In addition, dairy is a rich source of vitamin D, which is necessary for calcium absorption and bone health. Furthermore, dairy products are low in sodium, which can help maintain healthy blood pressure and reduce the risk of developing kidney disease.

One potential pathway is indirect; that is, dairy components influence other physiological functions that directly affect renal function [1,2]. Currently, hypertension and T2D are the leading causes of CKD. Elevated blood pressure damages the blood vessels and the nephrons in the kidneys, weakening them and their ability to filter out waste products. Elevated blood sugar levels also have a similar effect; by forcing the kidneys to work harder, T2D weakens the kidneys to the point where they pass nutrients the body needs (such as the protein albumin) into urine along with waste products [7]. Indeed, increased albumin in urine, a condition referred to as albuminuria, is itself diagnostic of CKD.

Consumption of dairy foods has been linked to a reduced risk for both hypertension and T2D [9-13]. By keeping blood pressure and blood sugar in check, it stands to reason that dairy also may modify the risk for developing CKD. Indeed, diets designed to lower blood pressure have also demonstrated a protective effect for CKD [3-5]. The DASH diet (Dietary Approaches to Stop Hypertension) focuses on eight food components: high intakes of fruit, vegetables, nuts, legumes, and low-fat dairy combined with low intakes of sodium, sweetened beverages, and red/processed meats [3]. The health benefits conferred by this diet on both blood pressure and cardiovascular disease are attributed to the additive effects of the food components. That is, it is not simply one food ingredient that improves vascular health but the synergistic actions of all ingredients [3]. Viewed this way, dairy foods are part of a larger dietary strategy to improve health including kidney disease but are not themselves the focus of the studies. Nevertheless, a 23-year study of nearly 15,000 middle-aged American men and women [3] identified a lower risk for kidney disease in participants with a higher dairy product intake. Legume and nut intake also demonstrated a protective effect against CKD, whereas consumption of red/processed meat increased CKD risk [3]. These results were echoed in a six-year study on 1600 Middle Eastern adults [4]. Study participants that did better at sticking to the DASH diet, including higher consumption of dairy foods, had lower odds for developing CKD. In both DASH diet studies [3,4], lower CKD risk was not only attributed to what the participants were eating more of but also what they were eating less of (i.e., sodium, red meat, and sugary drinks). Thus, it is not possible to say whether low-fat dairy foods would have had a protective effect (or as significant of an effect) in the absence of the other components of the DASH diet.

It also is possible that dairy foods directly influence the health of the kidneys through their anti-inflammatory and antioxidant...
Oxidative stress and inflammation are risk factors for CKD and for numerous other chronic diseases, including cancer, T2D, hypertension, and cardiovascular disease. Calcium, magnesium, vitamins A and E, dairy proteins, and even dairy fats have demonstrated antioxidant and anti-inflammatory properties. Thus, it could be multiple dairy ingredients, or the effect of their synergistic actions, influencing kidney function.

Support for a direct effect of dairy on the kidneys comes from a 2016 study [2] on nearly 4000 Dutch adults (aged 25–65) with normal or mildly decreased GFR. The metric of interest was changed in GFR over an extended period of time, and each subject had three or more examinations at five-year intervals from 1993 to 2012. The study authors found that a higher consumption (defined as ≥2 servings per day) of milk and low-fat dairy products was associated with a smaller decline in GFR per year. Because the study authors controlled for hypertension and T2D in their statistical analyses, the observed protective effect of dairy consumption, albeit minor (0.11 ml/min per year less decline), was attributed directly to the actions of dairy components, such as calcium, magnesium, and unsaturated fats [2].

One drawback of this study was that it did not look at the influence of dairy foods on kidney function in individuals diagnosed with CKD, making it difficult to say whether the actions of dairy ingredients influence kidney function in all individuals or only those with normal or slightly impaired kidney function. This issue was addressed, however, in a study [1] published just one month after the Dutch study [2]. Gopinath and colleagues [1] report on the association between dairy food consumption, calcium intake, and CKD over a ten-year study period in 1185 Australian adults living in the same area just west of Sydney. Baseline GFR values were not used for inclusion in the study, and thus these values varied across participants from within the normal range to diagnostic of CKD. Like the Dutch study [2], Gopinath et al. [1] found that consuming ≥2 servings of low-fat dairy foods per day was associated with a reduced prevalence and incidence of CKD during the study period, independent of hypertension and T2D. This study also compared calcium intake to CKD prevalence and found that individuals who had more calcium in their diet were less likely to have CKD over the study period [1]. Thus, the observed protective effect of low-fat dairy foods is at least partly due to the anti-inflammatory and antioxidant properties of calcium. In the same study population, poor calcium intake was associated with abnormal blood vessels in the retina, and the authors hypothesize that a similar physiological response may be taking place in the kidneys, wherein calcium influences renal blood vessel health [1]. Importantly, dairy and (calcium) intakes were below the recommended daily intake values for 83% of the participants [1], suggesting that even moderate calcium consumption can influence kidney function.

As observational studies, neither the Dutch [2] nor the Australian [1] study was able to establish a cause-and-effect relationship between dairy components and kidney health. But when these studies are combined with numerous others that indicate a protective effect of dairy food consumption on the development of chronic diseases [9–13], it provides a strong framework for developing more rigorous controlled studies. With the rapid increase in the incidence of CKD and other chronic diseases, it is encouraging that easy-to-implement lifestyle changes, such as eating two or more servings of low-fat dairy foods per day, could have such profound health benefits.


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Human Milk Oligosaccharides Alter Immune Cell Populations in Pigs

- A new study examines the effects of human milk oligosaccharides (HMOs) and prebiotic oligosaccharides on immune cell populations in uninfected and rotavirus-infected piglets.
- The study found that dietary HMOs altered systemic and gastrointestinal immune cell populations in piglets, and had a greater effect on immune cells than prebiotic oligosaccharides.
- Dietary HMOs were previously shown to have some effects on rotavirus infection susceptibility, and the study suggests that HMO-associated changes to immune cell populations may mediate some of these effects.

Rotavirus is a major viral pathogen, and rotavirus-associated diarrhea is prevalent in many developing countries [1-4]. Interestingly, breastfed infants have a lower incidence of rotavirus infection than formula-fed infants, suggesting that certain components of human milk may have protective effects against this virus [5].

“Human milk is best, you’re going to have the best protection if you’re able to feed your baby human milk,” says Professor Sarah Comstock of Michigan State University. “We’re always interested in how we can improve the immune responses of formula-fed infants to make them more like those of human-milk fed infants,” she says.

Researchers have investigated whether sugars found in human milk, called human milk oligosaccharides (HMOs), might be responsible for some of the protective effects against rotavirus. HMOs are absent from most infant formulas and have antimicrobial and immunomodulatory actions in vitro [6-8].

In a new study conducted by Comstock and Professor Sharon Donovan of the University of Illinois, the researchers measured the effects of HMOs on immune cell populations from uninfected and rotavirus-infected pigs [9]. They also compared the effects of HMOs with those of prebiotic oligosaccharides. “We wanted that comparison group in there because prebiotics were already being added to infant formula, and it doesn’t make much sense to spend a lot of effort and money adding HMOs to formula if prebiotics can be just as effective,” says Comstock.

The researchers found that dietary HMOs altered systemic and gastrointestinal immune cell populations in pigs, and had a greater effect on immune cells than did prebiotics. The findings suggest that the HMO-associated change in immune cell populations may mediate protective effects of HMOs on rotavirus infection. “It’s just more evidence that breastfeeding really is good for your baby,” says Comstock.

Researchers have long been interested in studying the effects of HMOs on the immune system, but it’s only recently that they have been able to cost-effectively conduct such studies. “Up until the mid-2000s, even though there was an interest in the human milk oligosaccharides, it was really hard to cost-effectively synthesize them or to get access to enough HMOs to do these types of experiments,” says Comstock. “It finally seemed feasible to do this type of study, to actually feed a significant amount of human milk oligosaccharides to piglets and look at how it affected their response to infection,” she says.

Comstock and Donovan decided to test the effects of HMOs on rotavirus infection in a piglet model, whose immune and gastrointestinal systems share many similarities with those of humans. “When you combine the immune homology with this gastrointestinal homology, and you’re looking at a gastrointestinal virus, I think it’s just really the best system,” says Comstock. “The payoff in terms of using this type of model, which is so much closer to a human than using a rodent model, is huge,” she says. “I think we’ll begin to see more work in humans and I think you’ll see that the piglet research really is an important driver of what we might expect in those human studies,” says Comstock.

The new study builds on several previous studies by Comstock and Donovan looking at the interaction of HMOs, rotavirus and the immune system [7, 8, 10]. “We knew in vitro that HMOs were able to inhibit rotavirus infectivity,” says Comstock. “We had a lot of evidence that there were direct interactions of HMOs with rotavirus, which were preventing rotavirus from getting access to the intestinal epithelium,” she says. “Then we had evidence that feeding HMOs would definitely affect the types of microbes that were living in the intestine, and we additionally had evidence that HMOs could directly affect immune cells ex vivo,” says Comstock. “We wanted to put this all together and ask, are these effects happening in vivo,” she says.

In the new study, Comstock and her colleagues compared the immune cell populations of uninfected and rotavirus-infected pigs fed either a control formula, a mix of HMOs consisting of 2'-fucosyllactose, lacto-N-neotetraose, 6'-sialyllactose, 3'-sialyllactose, and free sialic acid, or prebiotics consisting of short-chain galactooligosaccharides and long-chain fructooligosaccharides.

Both infected and uninfected HMO-fed pigs had increased peripheral blood mononuclear cell natural killer cells and mesenteric lymph node memory effector T cells compared with pigs fed formula. The researchers also found that dietary prebiotics induced intermediate increases in immune cell populations compared with dietary HMOs. Prebiotic oligosaccharides may be less effective than HMOs because of differences in their structures or in the intestinal bacteria they affect.
The researchers concluded that dietary HMOs were more effective than prebiotics in altering systemic and gastrointestinal immune cells in pigs. Comstock suggests that the HMO-associated changes to immune cell populations may mediate the effects of dietary HMOs on rotavirus infection susceptibility.

Comstock notes that the current study only looked at the effects of 5 HMO structures out of the more than 200 HMOs identified so far. “Even from this limited complement of HMO structures, we’re able to get increased immune responsiveness, so imagine what it might be like if you were able to include a full complement of these 200-plus structures,” she says.

“Obviously it would be nice if we could get all 200 into infant formula; the more we can make infant formula look structurally like human milk, I think the better off those babies’ immune systems will be,” says Comstock. “Unfortunately, a lot of those are even more expensive or impossible to synthesize or extract,” she says.

Follow-up studies could look at the immune effects of HMOs in more detail, including analyzing their effects at different post-infection time points. “One limitation of this study is that we had to pick one time point to look at these effects,” says Comstock. “That does limit our knowledge in terms of the full complement of effects that HMOs can have on the immune response,” she says.

The researchers plan to further analyze their data to try to better understand how the HMOs affect rotavirus-infected piglets. Understanding the in vivo effects of HMOs could help researchers design improved infant formulas and find ways to protect against or treat rotavirus infection. “We do plan to do more detailed and sophisticated modeling of our datasets to see if we can gain any knowledge or generate any hypotheses about how the bacterial changes and the immune system changes are working together to resolve the infection and alter the clinical response,” says Comstock. “The question is, can we model those interactions to understand more fully what’s going on to reduce the length of diarrhea in the rotavirus-challenged piglets,” she says.

Future studies could also try to decipher the mechanism by which HMOs influence immune cell populations. “It would be great to really understand the mechanisms of what is happening,” says Comstock. “We don’t know if these HMOs are directly interacting with immune cell receptors and directly triggering these changes in the immune cells, or if through interactions with bacteria or viruses in the intestine they’re modulating the extent of the immune response that needs to happen,” she says.

“So there’s still a black box between feeding of the HMOs and the clinical response, but this study gives us some clues about what might be going on” says Comstock.


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Holder Pasteurization Has Limited Impact on the Nutrients in Human Milk

- Holder pasteurization is the standard treatment for human milk donated to milk banks across the world.
- The evidence suggests that Holder pasteurization reduces the amounts of several vitamins in milk, and may alter the digestibility of certain proteins.
- Although the carbohydrate concentrations in human milk appear unaffected by Holder pasteurization and freezer storage, some studies suggest that fats are slightly decreased, and may be even more so if they stick to the plastic equipment that is often used to feed young infants.

Holder pasteurization, or HoP, is used the world over to help ensure that the milk distributed by human milk banks is safe for infants to consume. Thanks to its broad effectiveness at destroying a long list of bacteria and viruses—including HIV and Ebola—HoP is
recommended by the World Health Organization and the American Academy of Pediatrics. But does raising the temperature of human milk to 62.5°C for half an hour break down any of its constituents such that the nutritional content of milk is affected? This second article in a five-part series about HoP finds that the evidence on this topic is relatively thin: different methods of evaluating the composition of milk have frequently led to different conclusions. Overall, however, even though some studies indicate that several vitamins, iron, fats, and certain proteins can be altered by HoP, researchers rarely consider the nutritional changes to be clinically relevant.

There are several micronutrients for which evidence of a change resulting from HoP is unequivocal. Vitamin C, for example, has repeatedly been shown to be destroyed by HoP, whereas vitamins A and E are thought to be better retained [1]. But the data are less clear for some other vitamins. For example, several studies show HoP-induced reductions in the B vitamins, while others do not [1].

Last year, Fabio Gomes of the University of Queensland, in Brisbane, Australia, and his colleagues, used liquid chromatography and mass spectrometry to analyze the concentrations of vitamin D compounds in human milk before and after pasteurization [2]. They found that various vitamin D compounds were affected—vitamin D2, D3, 25(OH)D2 and 25(OH)D3—with losses due to pasteurization in the 10%—20% range. Whether this could have any impact on infant health is little understood: while vitamin D’s importance in the regulation calcium and phosphate absorption in the intestines is well established, the vitamin D requirements of pre-term infants, who often receive milk-bank milk, are not.

Some of those same Brisbane-based scientists also contributed last year to a paper about the impact of HoP on the mineral content of milk [3]. Again using mass spectrometry, they found no evidence for alterations in the amount of the trace elements zinc, copper, selenium, manganese, iodine, molybdenum, and bromine. The iron content of milk did decrease by 6.5% after HoP—a statistically significant shift—but this loss is not thought to be problematic for premature infants. Authors Nor Mohd-Taufek and her colleagues do not recommend milk supplementation with iron as a matter of course.

Sugars in milk appear to hold up well to heat treatment and storage. This is the conclusion of a recent review that evaluated the evidence around changes in the amounts of lactose and glucose—as well as glycans such as glycosaminoglycans and oligosaccharides [1]. The summary of 44 papers, which all measured changes in human milk’s composition before and after pasteurization, was written by the University of Turin’s Chiara Peila, and a team that is spread among various Italian institutions. The concentrations of the vast majority of the carbohydrates investigated did not change. When it came to glucose, however, different studies have found its concentrations to increase, to decrease and to remain constant after HoP.

Fats in milk store much of the energy that powers infant wriggling, smiling and crying. Crucially, they also help premature infants put on weight. Hence any reduction in the fat content of donor milk could have important consequences for choices around milk supplementation. (As it is, mothers who donate milk have generally been lactating for a while, and as a result, produce milk with a lower energy content than newer mothers [4].)

Two studies have addressed the question of whether HoP alters human milk’s fat content, with one by Ley et al. [5], which relied on bomb calorimetry, reporting no change, and another by García-Lara et al. [6] noting a decrease. García-Lara’s team used infrared spectrometry to analyze 34 samples of donated milk from 28 donors to the Hospital 12 de Octubre in Madrid, Spain. They report that pasteurization followed by freezer storage for 180 days resulted in 6.2% decrease in fat and a 5% decrease in the energy content of milk, respectively. Of the 6.2% decrease, 3.5% was the result from HoP, and the remainder (2.7%) was due to storage. The argument that the process of freezing and thawing might influence pasteurized human milk’s fat content is also backed up by work by Alan Araujo Vieira and his colleagues at the Fernandes Figueira Institute, in Rio de Janeiro, Brazil [4]. They report a 5.5% reduction in human milk’s fat content following pasteurization and a huge further reduction of 56.6% as a result of continuous infusion delivery (one of the two main ways of delivering milk to infants who cannot suck). Vieira and her colleagues suggest the explanation for such a large drop might be rupture of fat globule membranes that could facilitate fats sticking to the walls of the plastic equipment and syringes that are commonly used to feed very young infants.

Research into the impact of HoP on the proteins in milk tends to focus on changes in the proteins’ levels of activity, such as in the immunological functions of some proteins, rather than on the amount of protein available as a nutrient. There are good reasons for this, which will be explored in next month’s issue of SPLASH!®. Perhaps the most in-depth study on the topic, however, combines these two concerns.

In this sense, Cristina Baro at CNR’s Institute of the Science of Food Production, in Turin, Italy, and her team, note that the digestibility (and thus the nutritional supply) of the proteins in human milk can be reduced when some amino acids undergo carbonylation [7]. So they approached the question of the how much nutritionally available protein is present before and after HoP by immunostaining the protein component of human milk with a compound that binds to carbonyl groups. When they compared the carbonylation of one of the most important proteins in milk, β-casein, before and after HoP, they found it little changed (but caseins...
were somewhat degraded by the heat treatment). Another important protein, lactoferrin, did show considerable difference, however. Because lactoferrin binds iron, its carbonylation during HoP may explain the small reduction in human milk’s iron content that Mohd-Taufek and her colleagues reported last year [3].

Lactoferrin is much more than a building block to be cut up by gut enzymes and reconstituted in the form of infant muscle. It can itself fight germs and is thought to even stimulate infant intestinal development. Check back next month to learn more about how this, and other biologically active proteins in human milk, are affected by HoP.


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Human Milk Oligosaccharides Can Directly Inhibit Streptococcus Growth

- A new study finds that human milk oligosaccharides (HMOs) can directly inhibit the growth of group B Streptococcus (GBS).
- GBS growth is inhibited by certain neutral, non-sialylated forms of HMOs, and appears to be mediated by a GBS-specific glycosyltransferase.
- The neutral HMOs act synergistically with common antibiotics, and the results suggest that HMOs could potentially be used as direct antimicrobial therapies.

Sugars found in human milk, called human milk oligosaccharides (HMOs), have various protective effects against infectious agents [1,2]. HMOs are known to prevent the attachment of microbial pathogens to the host [3-5]. They also have other protective effects against infections by acting on the host immune system [6,7].

In a new study, Professor Lars Bode of the UC San Diego School of Medicine and the Larsson-Rosenquist Foundation Mother-Milk-Infant Center of Research Excellence found that HMOs can directly inhibit the growth of group B Streptococcus (GBS) [8]. “This is yet again a completely new direction to the story. The HMOs are acting not by serving as anti-adhesives, not by having an effect on the host, but by directly doing something to the pathogen,” says Bode.

The results of this study further add to our understanding of the antimicrobial benefits of human milk. “The number one lesson is that human milk is a very powerful tool for fighting infections, and yet another reason to empower women to breastfeed if they can,” says Bode.

Bode and Ann Lin, the first author of the current study, had previously shown that HMOs regulate the host innate immune response in order to prevent uropathogenic Escherichia coli (UPEC) invasion into bladder epithelial cells [9]. “The oligosaccharides reprogram epithelial cells and have a protective effect on the host that makes the host safer against the pathogen,” says Bode. But in that study, HMOs did not have any direct effect on bacterial growth.

After that study, Lin decided to look at the effects of HMOs on various other pathogens. She obtained several different pathogens from co-author and collaborator Professor Victor Nizet. “She sprinkled HMOs on a variety of pathogens, and all of a sudden found that group B Streptococcus would just not grow anymore,” says Bode. “Even in the complete absence of host cells, the HMOs had a direct effect on the pathogen,” he says. “This was completely new,” says Bode.

The HMOs inhibited the growth of GBS, but did not kill the pathogen even at high concentrations. They did not inhibit the growth of UPEC, Pseudomonas aeruginosa, or methicillin-resistant Staphylococcus aureus.
For their initial experiments, the researchers used HMOs isolated from pooled human milk. “We always start out with oligosaccharides isolated from pooled donor milk from different moms, as every mom makes slightly different oligosaccharides,” says Bode. The researchers then set out to identify the specific HMOs involved in inhibiting GBS growth. “We have about 150 to 200 different oligosaccharides, so the question is, which one is responsible?”

The researchers used multidimensional chromatography to separate the pooled HMOs into various fractions, and found that the inhibition of GBS growth was restricted to a fraction of non-sialylated, neutral HMOs. Interestingly, this is in contrast to the group’s previous findings, where it was sialylated HMOs that influenced host innate immune defense against UPEC. “We have 150 to 200 oligosaccharides, and if it was the same two or three that were responsible for all these effects, we wouldn’t need so many,” says Bode. “At this point it’s more of a coincidence if we find the same oligosaccharide doing things over and over,” he says.

Based on their analysis of the neutral HMO fraction, the researchers selected nine commercially available oligosaccharides to test. The researchers found that GBS growth was inhibited by lacto-N-tetraose (LNT) and lacto-N-fucopentaose I (LNFP-I). Interestingly, a structural isomer of LNT called lacto-N-neotetraose did not significantly inhibit GBS growth. “What really surprised us was that lacto-N-neotetraose doesn’t work,” says Bode. “A very tiny change, just a little bit of the structure attached slightly differently seems to make a big difference,” he says.

To elucidate the mechanisms by which the HMOs might be inhibiting GBS growth, the researchers then screened a library of GBS mutants that they obtained from co-author Professor Kelly Doran. “One of the mutants didn’t respond to the oligosaccharides, it grew just as well with and without oligosaccharides,” says Bode. “That gave us the idea that the protein encoded by the mutated gene could be a potential target for the effect of the HMOs,” he says.

Through bioinformatic analysis, the researchers identified the mutated gene as a putative glycosyltransferase, and confirmed through directed mutations that this gene was responsible for the susceptibility of GBS to neutral HMOs. “Our thinking is that this glycosyltransferase takes components of the HMOs and incorporates them into the cell wall structures that then can no longer be elongated and won’t allow the bacteria to grow,” says Bode. The researchers are currently working on labeling individual oligosaccharides to see which gets incorporated in the cell wall.

This particular glycosyltransferase also appears to be unique to GBS, which could explain why HMOs did not inhibit growth in the other bacteria tested. “If bacteria don’t rely on this glycosyltransferase, then they won’t put HMOs in the cell wall, and can continue to grow,” says Bode.

Bode and his colleagues also found that the neutral HMOs acted synergistically with common antibiotics, as prior exposure to HMOs dramatically reduced the inhibitory dose of vancomycin and ciprofloxacin. “That was probably the coolest part of the story, to find synergistic effects,” says Bode. “Given the crisis of antibiotic resistance, it could certainly help to be able to use a lower antibiotic dose in the presence of HMOs,” he says.

HMOs could also have additional therapeutic utility besides being used in conjunction with existing antibiotics. “I’m thinking ahead to being able to develop these as novel drugs,” says Bode. “They would be a very attractive addition to our arsenal of antimicrobials,” he says. “We would expect HMOs to pass relatively easily through regulatory requirements, since we feed them to babies naturally all the time,” says Bode. “It would be a great application of a natural compound,” he says.

There’s still some ways to go before researchers confirm whether HMOs could have therapeutic potential against GBS in humans. “It’s great to see this in a test tube, but we eventually want to see if this effect translates in humans, especially now that we’re starting to get FDA GRAS-approved HMOs,” says Bode. “[GRAS” is an acronym for Generally Recognized As Safe, an FDA designation referring to a substance or chemical added to food that is considered by qualified experts to be safe under the conditions of its intended use]. “There’s still a lot of work to be done, but it’s very exciting, it’s a great field to be in,” he says.


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